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BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES

Paper No. 35

Application Number: 09/408,905
Filing Date: September 29, 1999
Appellant(s): WALSH, KENNETH

Helen C. Lockhart
For Appellant

EXAMINER'S ANSWER

This is in response to the appeal brief filed August 15, 2003.

(1) Real Party in Interest

A statement identifying the real party in interest is contained in the brief.

(2) Related Appeals and Interferences

A statement identifying the related appeals and interferences which will directly affect or be directly affected by or have a bearing on the decision in the pending appeal is contained in the brief.

(3) Status of Claims

The statement of the status of the claims contained in the brief is correct. However, it is noted that that the *listing* of the Claims (Item B, top of page 2) is incorrect. Claims 1-4 are rejected.

(4) Status of Amendments After Final

The appellant's statement of the status of amendments after final rejection contained in the brief is correct.

(5) Summary of Invention

The summary of invention contained in the brief is correct.

(6) Issues

The appellant's statement of the issues in the brief is correct.

(7) Grouping of Claims

Applicant's statement that the claims stand or fall together is substantially correct. It is noted that this does not include "all claims" but rather the claims on appeal, Claims 1-4.

(8) Claims Appealed

A substantially correct copy of appealed claims 1-4 appears on page 13 of the Appendix to the appellant's brief. The minor errors are as follows: Claim 5, although listed in the Appendix, is not under appeal.

(9) Prior Art of Record

Cuevas *et al.*, "Fibroblast growth factor-1 prevents myocardial apoptosis triggered by ischemia reperfusion injury" Eur. J. Med. Res. 1997, Nov 28; 2(11):465-8.

Datta *et al.* "Akt phosphorylation of BAD couples survival signals to the cell-instrinsic death machinery". Cell. 1997 Oct 17; 91(2):231-41.

(10) Grounds of Rejection

The following ground(s) of rejection are applicable to the appealed claims:

Claims 1-4 are rejected under 35 U.S.C. 103(a) as being unpatentable over Cuevas et al. (Eur.J.Med.Res, Vol. 2, pages 465-468, November, 1997) in view of Datta et al. (Cell, Vol. 91, pages 231-241, October, 1997) as originally set forth in Paper No. 13.

(11) Response to Argument

Applicants argue (page 5) that one of skill in the art would not have combined the Datta *et al.* and Cuevas *et al.* references because the components of the reference are not simply interchangeable. Applicants argue (top of page 6) that FGF and Akt are distinct molecules wherein applicants argue that "FGF does not activate Akt in cardiomyocytes, skeletal myocytes, or vascular endothelial cells". This argument has been considered but is not found relevant because it appears that applicants are attempting to refute the possibility that FGF may activate Akt. However, the idea that FGF may activate Akt was never set forth in the previous actions. Hence, it does not appear that this argument is relevant.

Applicants further argue that one of skill in the art would not expect that Akt, an apoptosis inhibitor reported to be effective in neurons, would be effective in cardiac myoctes, in the absence of further scientific evidence. This argument has been considered but is not found persuasive. Although the Datta *et al.* reference does not specifically include cardiac cells as a particular cell type where Akt would inhibit apoptosis, the reference teaches (page 231, 2nd column, 3rd paragraph) that Akt has been shown to suppress the apoptotic death of a number of cell types (including lymphoid, neuronal, fibroblast, and epithelial cells - see Paper No. 30, page 4 as so indicated by applicants) induced by a variety of stimuli. Applicants counter (page 7) the latter by arguing that that none of the references cited by Datta *et al.* teach or suggest the expression of Akt in cardiac myocytes. This argument has been considered but is not found persuasive. As set forth above (and in Paper No. 32) the prior art suggests a reasonable

expectation of success that Akt would inhibit apoptosis in a variety of cell types including cardiac myocytes. Further, one of the references that has been cited by Datta *et al.* suggests that Akt is rather ubiquitous in that Ras transformation activates the Akt cellular survival pathway (Khwaja *et al.*). Thus, because the Ras protein is well known in the art to be present in a wide variety of different cell types, the title of this reference reasonably suggests to one of ordinary skill that Akt would also be present in a wide variety of cell types in order to facilitate their interactions.

Applicants further argue (pages 8-9) that there would be no reasonable expectation of success for combining the Datta *et al.* reference and the Cuevas *et al.* references because there is more than one apoptotic pathway and not all apoptosis inhibitors are alike. This argument has been considered but is not found persuasive because despite the alleged "different pathways", both molecules function to inhibit cell death. Whether or not they achieve this by different pathways is irrelevant. Furthermore, arguments that rely on a particular distinguishing features are not persuasive when those features are not recited in the claims.

Applicants further argue (page 10) that the Examiner relied on impermissible use of hindsight to arrive at the rejection. Applicants argue that without Appellant's disclosure the Examiner can point to no teaching in the prior art that would motivate one of ordinary skill in the art for such a teaching. Applicants further argue (page 11) that based on this rationale, one would necessarily conclude that one skilled in the art would have been motivated to substitute any apoptosis inhibit for FGF-1 for treating <u>any</u> disease which involved apoptotic cell death. This argument has been considered but is not found persuasive. The rejection was solely based on knowledge that was within the level of ordinary skill in the art at time the claimed invention was

made and does not include knowledge gleaned from applicant's disclosure. All that is required is a reasonable expectation of success. In this case, the art teaches that Akt and FGF-1 are functionally equivalent inhibitors of apoptosis. For the latter, it was well known in the art at the time the invention was made that FGF-1 would predictably treat a myocardial infarction by inhibiting the necrosis of cardiac tissue. Indeed the conclusion by Cuevas *et al.* recites (abstract) that the programmed myocyte cell death (e.g. apoptosis) triggered by ischemia-reperfusion is attenuated by FGF-1. Akt, also a protein, was well known in the art at the time the invention was made to be an effective inhibitor of apoptosis in *a variety of cell types*. The fact that the reference does not specifically teach one of those cell types to be cardiac myocytes does not constitute a teaching away from the broader disclosure of cell types in general. It is perfectly reasonable to expect that Akt would successfully inhibit apoptosis in a variety of cell types, *including* cardiac myocytes. Hence, the teachings of both references taken together, suggest the claimed invention.

For the above reasons, it is believed that the rejections should be sustained.

Respectfully submitted,

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Examiner
Art Unit 1642

GBN
December 1, 2003

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